Original Article

Major Components of Metabolic Parameters and Nutritional Intakes in Different Genotypes of Adiponectin +276 G>T Gene Polymorphism in Non-Diabetes and Non-Alcoholic Iranian Fatty Liver Patients

Fatemeh Mohseni, Sahar Moghbelinejad *, and Reza Najafipour

Cellular and Molecular Research Center of Qazvin University of Medical Science, Qazvin, Iran

Abstract

Background: Genetic and environmental factors are both involved in the etiology of Non-Alcoholic Fatty Liver Disease (NAFLD). Among the genetic factors, certain polymorphisms of adiponectin gene are associated with NAFLD. In the current study, we investigated the association between metabolic parameters with different genotypes of adiponectin +276 G>T polymorphism among the Iranian NAFLD patients, and the effect of nutritional intake with development of NAFLD.

Methods: In this study, 75 patients with NAFLD and 76 healthy individuals were enrolled. Dietary intakes were assessed using a semi- quantitative Food-Frequency Questionnaire (FFQ). Body Mass Index (BMI) and Waist to Hip Ratio (WHR) were calculated. Biochemical assays including FSG (Fasting Serum Glucose), liver enzymes, lipid profiles, Malondialdehyde, insulin resistance and Total Antioxidant Capacity (TAC) were measured after 12 *hr* fasting. Gene polymorphism study was done by using of sequencing method.

Results: Although, T allele frequency was more prevalent in patients with NAFLD than control, adiponectin +276 G>T polymorphism was not associated with risk of NAFLD. Among the metabolic parameters, TAC in TT genotype was significantly lower 1.44(0.69 to 2.81) p>0.05, AST in GT, GG genotypes, and ALT in all three genotypes were higher in NAFLD patients in compared to healthy subjects (p<0.05). Patients with GT genotype have significantly lower fat consumption and vitamin E intake as compared to control group with the same genotype (p<0.05).

Conclusion: In this study, we showed the association of different genotypes of +276 G>T polymorphism in adiponectin gene with some metabolic parameters.

*** Corresponding author:**
Sahar Moghbelinejad, Ph.D.,
Cellular and Molecular Research Centre, Qazvin University of Medical Sciences, Qazvin, Iran
Tel: +98 2813336001
Fax: +98 2813324970
E-mail:
smoghbelinejad@qums.ac.ir
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Introduction

Non-Alcoholic Fatty Liver Disease (NAFLD) is one of the most common hepatic disorders with unusual lipid deposition in hepatocytes [1]. NAFLD is an epidemic metabolic liver disease observed in many countries and its prevalence is increasing worldwide [2]. According to the US National Health and Nutrition Examination Survey (NHANES), the prevalence of NAFLD among chronic liver diseases grew from 47 to 75% between 1988 and 2008 [1]. The prevalence of NAFLD in the adult general population was reported 21.5% in a large population-based study in southern regions of Iran in 2011 [3].

NAFLD is the hepatic manifestation of the metabolic syndrome because obesity and insulin resistance are in close inter-relationship with NAFLD [1,4]. Numerous studies have suggested that insulin resistance is characteristic of NAFLD [5]. Insulin resistant patients with NAFLD show decreased insulin sensitivity either at the level of muscle or at the level of liver and adipose tissue [6,7]. Insulin resistance is related to increase of Free Fatty Acids (FFAs) flux that increases TG production and secretion of Very Low-Density Lipoprotein (VLDL) is stimulated in hepatocytes. Fat accumulation in liver is linked with lipid peroxidation and oxidative stress.[5] Oxidative stress phenomenon induces imbalance in the pro-oxidant/antioxidant equilibrium; a condition that may influence a number of pathophysiological events in the liver [8].

Genetic and environmental factors are both involved in etiology of NAFLD as a multifactorial disease; genetic polymorphisms and dietary intake have been identified as influencing factors in NAFLD development [9]. The long-term excessive intake of dietary composition in food groups is associated with NAFLD pro-